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DIE-BACK OF CHILLIES (*CAPSICUM* SPP.) IN BIHAR

BY

JEHANGIR FARDUNJI DASTUR, M.Sc.

Offg. Second Imperial Mycologist

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BY

JEHANGIR FARDUNJI DASTUR, M.S.,

Offg. Second Imperial Mycologist.

[Received for publication on 23rd April, 1921.]

THE most serious disease of chillies (*Capsicum annuum* and *C. frutescens*) in Bihar is the die-back disease, due to *Vermicularia Capsici* Syd., which causes considerable damage to the crop, in years when there is continuous rain or high humidity in the latter half of September and beginning of October. In Bihar, the disease first appears in the end of September or in the first half of October, when the plants are mature and have commenced to flower. It spreads virulently from field to field. In severe cases of attack the plants are either completely killed or so badly diseased that the yield of healthy fruits is negligible. The first nip of the dry cold weather puts a sudden check to the progress of the disease which eventually dies away; the plants then recover and put forth new healthy shoots. The critical period when the plants are subject to the attack of the disease is, therefore, of a short duration, about four to six weeks.

Plants growing under shade have been observed to suffer very little from this disease. Late sown crops are also very little affected but unfortunately yield a very poor return. Fruits that mature before the beginning of December get badly diseased, to the extent of about 35 per cent., but those that ripen later escape the disease, the percentage of infected fruits after the middle of this month being negligible.

Macroscopic characters.

(*a*) *The stem.* The attack as a rule commences from the growing point or the flower-bud, and therefore the presence of the disease in the early stage of attack is marked by the top of the affected branches withering and turning brown (Plate I, fig. 2). The plant dies back as the attack spreads downwards;

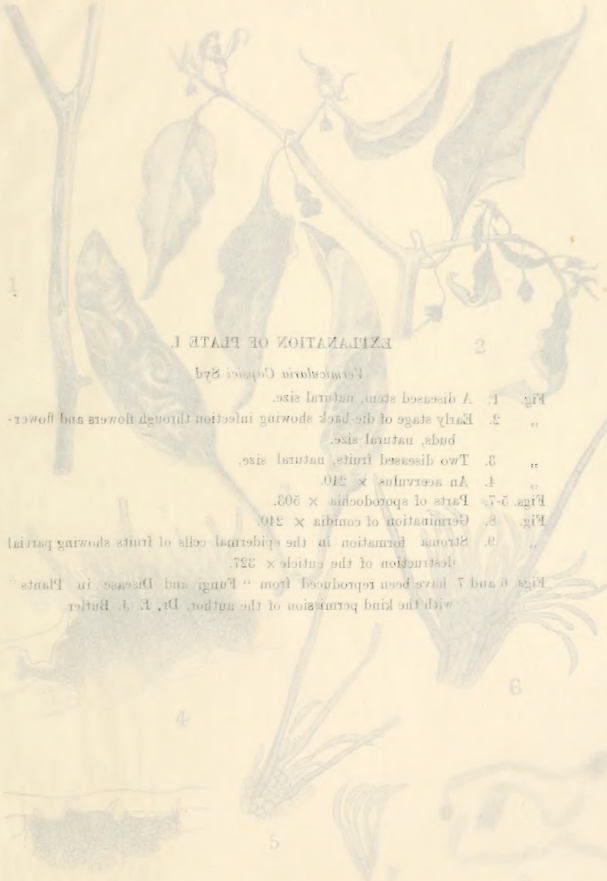
when it reaches a fork the infection runs up the sound limb. In some cases the attack starts not from the growing point but from a wound on the stem. As the disease progresses, the infected part of the stem takes on an enamelled white colour and is sharply demarcated from the healthy green bark by a black line running round the whitened area. The white of the diseased part is punctuated by scattered black bristly and minute elevations, which are the acervuli of the fungus (Plate I, fig. 1).

(*b*) *The fruit.* The fruits become visibly diseased when they turn red, very seldom while they are still green. The first outward sign of infection is the appearance of a small black circular speck, generally sharply defined but at times diffused. The disease spreads not concentrically, but more in the direction of the long axis of the pod, so that the originally circular spot becomes more or less elliptical. As the infection progresses the spot is either diffused and black or greenish-black or dirty grey, or is markedly delimited by a thick and sharp black outline enclosing a lighter black or straw-coloured area. Two or more diseased spots may become confluent, thereby destroying the regularity of the individual spots, but the delimiting black line is not always completely obliterated where the infected areas have united. Badly diseased pods lose their normal red colour and turn straw-coloured or in some cases pale white (Plate I, fig. 3). The acervuli are generally densely gregarious or scattered all over the infected parts ; at times they are concentric. They project a little above the surface of the pod and are bristly and carbonaceous. The spores ooze out of the acervuli in pink masses or strings under moist conditions.

When a diseased pod is cut open the lower surface of the skin is found covered with minute, black, spherical elevations ; these are the stromatic masses or sclerotia of the fungus. In advanced cases the seeds are covered by a felt of white mycelium, in which are embedded a few black or grey-green stromatic bodies. Infected seeds turn rusty in colour.

Microscopic characters.

(*a*) *The stem.* Sections through a slightly diseased stem show the infected tissues to have turned yellow or brown and the cell cavities to be filled with a similarly coloured gummy substance which hides the hyphæ. The use of clearing agents helps in disclosing them. The tissues outside the xylem are more readily discoloured and destroyed than the xylem itself. Diseased xylem tissues get discoloured, but not so much as the soft outer tissues ; and the presence of the hyphæ within the vessels can be clearly traced. Even in very advanced cases of attack, the xylem vessels have not been found choked



EXPLANATION OF PLATE I.

Verticillium Dieffii Syd.

Fig. 1. A diseased stem, natural size.

„ 2. Early stage of die-back showing infection through flowers and flower-buds, natural size.

„ 3. Two diseased fruits, natural size.

„ 4. An acervulus × 210.

Figs. 5-7. Pairs of sporodochia × 303.

Fig. 8. Germination of conidia × 210.

„ 9. Stroma formation in the epidermal cells of fruits showing partial destruction of the cuticle × 327.

Figs. 6 and 7 have been reproduced from "Fungi and Diseases in Plants" with the kind permission of the author, Dr. E. J. Butler.

EXPLANATION OF PLATE I.

Vermicularia Capsici Syd

Fig. 1. A diseased stem, natural size.

„ 2. Early stage of die-back showing infection through flowers and flower-buds, natural size.

„ 3. Two diseased fruits, natural size.

„ 4. An acervulus × 240.

Figs. 5-7. Parts of sporodochia × 503.

Fig. 8. Germination of conidia × 240.

„ 9. Stroma formation in the epidermal cells of fruits showing partial destruction of the cuticle × 327.

Figs. 6 and 7 have been reproduced from "Fungi and Disease in Plants" with the kind permission of the author, Dr. E. J. Butler.

VERMICULARIA CAPSICI SYD.

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with the mycelium as in the vascular wilts, and thus explains why a healthy leaf or pod is sometimes found attached to a stem completely ringed by the white diseased bark.

(*b*) *The fruit.* In the walls of the fruit the fungus overruns the soft tissues, destroying the cells. In the early stage of attack the cells are filled with a brown or yellow gummy substance which is not found at a later stage; the fungus also acts on the chromoplasts. The different colours of the diseased parts of the fruit are due to the quantity of the gummy substance present in the infected cells and to the extent to which the chromoplasts are altered. In the cells, a layer or two below the epidermis, the hyphæ commence to aggregate together, forming a loose mass of pseudo-parenchymatous cells, sub-hyaline or slightly brownish in colour. In the epidermal cells themselves a compact dark brown stroma of pseudo-parenchyma is laid down. Under the pressure of the growth of this stroma the cuticle is ruptured and lifted off the epidermal cells. The cuticle is not simply pushed up and ruptured by the fungus but is also partially eaten away (Plate I, fig. 9). On the surface of the brown stroma, now exposed to the air, a further development of lighter coloured fungus tissue, compo ed of elongated and narrow cells in almost parallel rows, occurs. The stroma of the acervulu is thus partly below the epidermis and partly above it (Plate I, fig. 4). The surface cells of the stroma become basidia (Plate I, fig. 6). The latter are hyaline, long and narrow. The conidia are hyaline, falcate, with an oil globule in the centre (Plate I, figs. 5—7). Setæ also arise from the pseudo-parenchymatous cells towards the surface of the stroma. They are abundant, simple, stiff, erect, septate and dark brown (fuliginous) in colour except at the tip which is lightly coloured. They occasionally bear conidia which are similar to those borne on the basidia (Plate I, fig. 5).

On the inside of the skin acervuli are not formed, but there are instead small round black or dark brown raised solid bodies composed of pseudo-parenchymatous cells formed by the conglomeration of hyphæ. These sclerotia-like bodies are also found on the thalamus.

(*c*) *The seed.* The inner walls of the normal cells of the seed coat are enormously thickened and finely striated. The outer wall is also thickened but very little in comparison with the other walls. This outer wall is composed of cellulose, while the inside walls contain both cellulose and lignin.*

* When treated with an acid solution of phloroglucin the inner walls of the seed coat show distinctly two layers. The centre of the thickened walls takes a red colour which shows the presence of lignin, while the rest of the thickened walls becomes light yellow in colour; but with Schulze's solution these parts stain blue, being composed of cellulose, and the centre stains yellow.

The fungus hyphæ enter the seed coat directly through the outer cellulose wall. The hypha, without necessarily forming an appressorium, bores its way through the upper wall and enters the cell cavity (Plate II, fig. 4). In badly infected seeds this upper wall has been very often found to be destroyed. The hypha now crosses the cell cavity and reaches the thickened inner walls. Here it swells at the point of contact, and from this swelling an exceedingly fine process is put forth which bores its way through, delignifying the wall (Plate II, fig. 5). As the result of delignification, cellulose is left. That this happens is clearly seen by the action of Schulze's solution; the tunnels in the thickened walls formed by the hyphæ take a blue colour with this reagent.

Thus, unlike the hyphæ of *Macrosporium Solani* Cke.,[1] which infect the tomato seed through the micropyle, the hyphæ of *Vermicularia Capsici* Syd. enter the perisperm and endosperm directly through the seed coat.

In the infected chilli seed there is no formation of a thick weft of compact mycelium between the seed coat and the endosperm, as found by Miss Massee in the case of the hybernating mycelium of *Macrosporium Solani* Cke. in tomato seeds. Even in advanced cases of attack the testa of the chilli seed is in contact with the inner tissues except where stromatic bodies are formed in the cells adjacent to the seed coat which is consequently slightly lifted up at this place. The hyphæ are septate, of variable thickness, inter- and intracellular, and hyaline, but they turn brown when they collect in masses to form stromatic bodies. These bodies are composed of small pseudoparenchymatous cells. They are found both in the cells of the seed coat and of the inner tissues. Some sections of diseased seeds have shown fruiting pustules embedded in the inner tissues.

An infected seed in which the embryo is not attacked will germinate; but conditions suitable for the germination of the seed are also favourable for the dormant stromatic bodies within the seed and on the seed coat to resume their activities; these stromatic bodies have been found to remain viable for at least a year. Therefore there is the danger of the embryo getting diseased at any stage of its germination as long as any part of it is within the seed coat. The infection may spread through the diseased endosperm or from the outside of the diseased seed coat to those parts of the germinating embryo in contact with them. If the infection takes place when the cotyledons are still completely

[1] Massee, J. On the Presence of Hybernating Mycelium of *Macrosporium Solani* Cke. in Tomato Seed. *Kew Bull.* 1911, No. 4, p. 145.

EXPLANATION OF PLATE II.

Vermicularia Capsici Syd.

Fig. 1. A hypha penetrating the cuticular layer of the epidermis of a fruit by means of a fine lateral projection × 624.

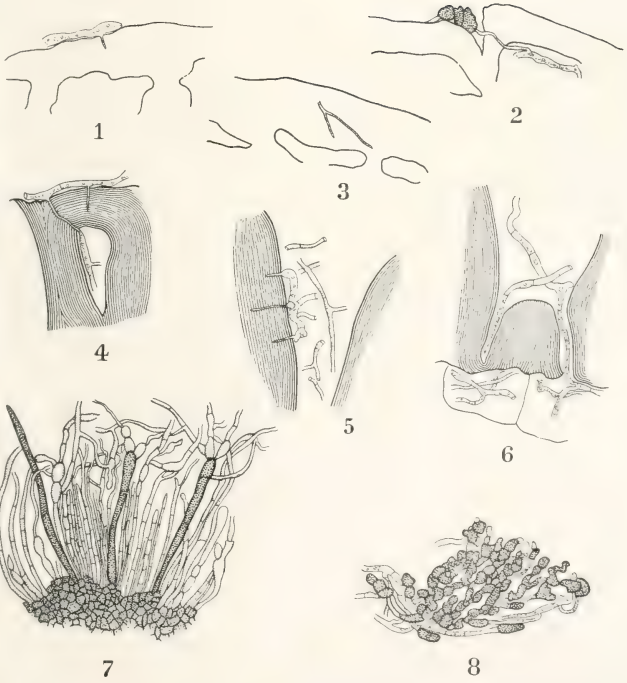
„ 2. A hypha from an appressorium penetrating the cuticular layer through a natural crack in the epidermis × 624.

„ 3 A branched hypha in the cuticular layer × 624.

Figs. 4-6. Hyphæ in the cells of the seed coat × 576.

Fig. 7 An acervulus with setæ forming a pseudo-pycnidium (early stage) × 266.

„ 8. A group of appressoria forming a stroma × 266.



VERMICULARIA CAPSICI SYD.

enclosed within the seed coat, the germinating embryo is killed almost at once, but if it spreads when only a part of the cotyledons are within the seed coat, the seedling may escape total destruction from the disease and may grow into a healthy plant, especially if the weather is dry enough to check the further progress of the disease.

Cultures of the parasite.

Shear and Wood[1] have found sterilized corn meal and corn-meal agar to be satisfactory media for growing the ascigerous stages of some fungi causing anthracnoses. Krüger[2] succeeded in getting the perfect stage of *Glœosporium musarum* on potato stems completely or partly sterilized.

Vermicularia Capsici has been cultivated many times on these media and on several others, but the perithecial stage has not as yet been obtained.

In agar media the early growth of the fungus is slightly ærial, white and floccose. As it grows it forms a thick leathery mat of interlacing hyaline hyphæ on the surface of the medium. In four or five days after inoculation black raised bodies like pin-heads are visible. These are either the acervuli or sclerotia of the fungus. A band of black or brown stromatic tissue is formed where the agar slant touches the glass slides. As the culture grows old, stromatic plates are laid down, closely in contact with the surface of the medium.

The ærial hyphæ are uniform in thickness and do not bear conidia like those of *Glœosporium piperatum*. The matted hyphæ on the surface of the medium and the hyphæ in its substance vary a great deal in breadth. Some are extremely fine, while others are very broad. The cytoplasm of the broad hyphæ, especially of those within the medium, is highly vacuolate. As the culture grows old, large hyaline vesicles appear as offshoots of the mycelium or short branches of the mycelium become very much enlarged at the tips. Daughter hyphæ occasionally arise through the septa from within the parent hypha. All hyphæ are hyaline except those that form acervuli, sclerotia and stromatic masses.

The acervuli in culture media appear as round black bristly heads. They are identical with those found on the host itself.

Pseudo-pycnidia. Acervuli, especially when old, are sometimes roofed over by a network of brown-coloured hyphæ formed by the profuse branching of some of the setæ and the marginal cells of the acervulus.

[1] Shear, C. L., and Wood, A. K. Studies on Fungous Parasites belonging to the Genus *Glomerella*. *U. S. Dept. Agr., Bur. Pl. Ind. Bull.* No. 252, p. 15, 1913.

[2] Krüger, F. Beiträge zur Kenntnis einiger *Glœosporien*, I and II. *Arb. Kais. Biol. Anst. f. Land-und Forstwirtschaft*, IX. Pt. 2, p. 251, 1913.

Sclerotia. These bodies are formed in two way : (1) Hyphæ collect together and form a solid black ball. On sectioning, this is found to be composed more or less uniformly of brown pseudo-parenchymatous cells, but in some cases there is a slight differentiation into cortex and medulla : the central cells are large and thin-walled and the peripheral cells have a smaller lumen and are thick-walled and deeply coloured. (2) At times what is potentially an acervulus is transformed into a sclerotium. As in the case of a normal acervulus, a base of brown-coloured pseudo-parenchyma is laid down. From this arise dark coloured setæ and parallel rows of light coloured thin and septate hyphæ which in the course of normal development would have borne basidia and spores ; interspersed with these fine hyphæ are found broad ones from which barrel-shaped cells are cut off. By the repeated branching of setæ and hyphæ and by formation of lateral outgrowths, a sclerotium is thus formed in place of an acervulus (Plate II, fig. 7). Sclerotia often have setæ like those of *V. carpini* Duc. on potato.

Stromatic masses. In old cultures on agar media stromatic plates are laid down closely appressed to the surface of the medium. These plates are composed of brown septate hyphæ. They seem to be filled with some reserve material. When hyphæ from the agar medium come in contact with the sides of the tube they bear appressoria which run into chains (Plate II, fig. 8). These chains unite into small triangular black or brown stromatic masses which creep up the sides of the tube and form a band round the margin of the medium.

On sterilized stems of *Capsicum* sp. and potato very little aërial growth is formed. Sclerotia and acervuli are produced in abundance.

When the acervulus becomes old and dry it is covered by a mucilaginous hard coat which keeps the enclosed mass of spores viable for several months but the conidia are very short lived if they get separated from the mass. Spores germinate in ordinary water by giving out germ-tubes from near their ends. When the germ-tube has grown a short distance there is formed at its end an appressorium. In some cases the germinating spore divides by a septum at the middle and may become constricted at this point (Plate I, fig. 8). The germ-tubes do not anastomose as those of *Gloeosporium piperatum* E. & E. They do not form secondary conidia on their tips or on lateral branches.

Sclerotia and stromatic masses may resume growth immediately under favourable conditions, but they are capable of retaining their vitality for several months. They "germinate" by giving out hyaline hyphæ from their cells.

Inoculation experiments.

All the inoculations were done with cultures originally started from a single spore.

Chillies. Seedlings grown in sterilized tubes containing moist cotton plugs from seeds removed aseptically from healthy chilli pods were inoculated either with spores or with hyphæ. The effect of the inoculations was noticed in three or four days by the inoculated parts turning brown. The infection spread rapidly and killed the seedlings in a couple of days ; *Vermicularia* acervuli were produced in abundance. The success of the inoculations and the spread of the infection depended upon the amount of moisture present in the tubes. If there was not sufficient moisture when the seedlings were inoculated, the inoculations did not take. If after the infections had started, the amount of moisture was reduced below a certain limit, the spread of the disease was checked.

Growing points and flowers of plants raised in pots were inoculated and kept under bell jars in the laboratory. The inoculations in some cases failed, and in others where the infection had started it did not make much progress ; but if the inoculated plants were kept in a moist atmosphere by covering the inside of the bell jars with moist blotting paper, the inoculations invariably succeeded and the infection spread rapidly, killing back the plant. The first signs of successful infection are the turning brown and rotting of the inoculated growing point or flower. As the infection spreads, the plant gradually dies back. Inoculations done on unwounded woody parts of the stem were unsuccessful ; wounded parts could be inoculated but not as readily and rapidly as the growing points and flowers.

Mature pods were readily infected but not the green fruits.

Microtomic sections of inoculated pods show that the hyphæ enter the skin directly or through natural cracks in the thick cuticle (Plate II, figs. 1 and 2) in the former case the hyphæ form an appressorium before entering the unbroken cuticle, unlike those attacking the seed coat ; a very fine process which penetrates the cuticle is produced from this appressorium. As long as the hypha is in the cuticle it remains exceedingly fine (Plate II, figs. 2 and 3) ; but it swells up to its normal size when it enters an epidermal cell (Plate II, fig. 2). It sometimes gets branched when within the cuticle.

Seeds of chillies have also been successfully inoculated. In the early stage of infection the seeds became rusty brown. Acervuli and sclerotia were formed on the seed coat and in the inner tissues as well under moist conditions.

Carica Papaya. Flowers of *Carica Papaya* have given successful inoculations. They showed in two days a brown, water-soaked appearance at the place of inoculation. *Vermicularia* acervuli were produced in four days; very young fruits could be inoculated either through wounds or through scars left by fallen petals. The beginning of the infection was marked by a circular depression at the inoculated part.

Vigna Catjang and *Dolichos lablab*. Pods of these vegetables did not take the inoculation so readily as flowers and young fruits of *Carica Papaya*. It was almost a week before the first symptoms of the infection were seen. The inoculated pods of *Vigna Catjang* became water-soaked while those of *Dolichos lablab* were depressed as the result of infection.

Solanum Melongena. Fruits of brinjal showed a slight browning in about ten days after inoculation which was done by wounding the epidermis. A little later the brown area became sunken and then black. The infected area was black in the centre and brown at the margins. Black acervuli were produced in the sunken areas and were arranged more or less concentrically.

Citrus sp. As the result of inoculation through a puncture a scabby growth was formed in a few days. This scab did not much increase in size and produced only a few scattered acervuli. Underneath this scab the tissues were rotting and had turned brown.

Inoculations on mango (*Mangifera indica*), plantains (*Musa* sp.), French bean (*Phaseolus vulgaris*), sweet peas (*Lathyrus odoratus*), onions (*Allium cepa*), sugarcane (*Saccharum officinarum*) and *Sorghum vulgare* have failed.

Treatment.

It was at first supposed that seed selection would perhaps play an important part in controlling this disease, but the desired result has not been obtained under field conditions.

Healthy and infected seeds were sown in two separate seed beds. The germination of the infected seeds was poor, about 60 to 70 per cent.; and 10 to 15 per cent. of the seedlings died of the disease before the cotyledons had completely opened. The germination of the healthy seeds was normal and not a single seedling was infected. Seedlings grown from infected seeds showed a slightly poorer growth than those raised from healthy seeds and this difference could be observed for some time after the seedlings were transplanted in two separate plots in a field; but it gradually wore off and at the time of flowering the plants in both the plots were equally vigorous. The field in which the plants were transplanted had never grown chillies before, and in fact was

brought under cultivation only the previous year. Nevertheless, the disease appeared in both the plots by the middle of October when the plants had commenced to set fruit, and was equally severe.

Treating infected seeds with different strengths of copper sulphate solution and of formalin before sowing was also tried, but naturally the rate of germination was not higher as the fungus has been found to hibernate within the seed.

During the chilli season of 1917 18 eight $\frac{1}{4}$-acre plots were laid under chillies to try remedial measures that had suggested themselves in the light of experience gained in previous years.

The writer wishes to make his acknowledgments to Mr. G. S. Henderson, Imperial Agriculturist, for the assistance he has given in carrying out these experiments. Acknowledgments are also due to Md. Taslim, Fieldman to the Imperial Mycologist, for the care he has taken in looking after these experiments.

The plan of the experiments is shown in the following diagram:

Bombay variety	Chillies grown with maize. One row of maize after every 3 of chillies	Control local varieties	Manured local varieties
1	2	3	4
Peshawar variety	Control local varieties	Local varieties sprayed with 1 p.c. Burgundy mixture	Manured local varieties late sown
5	6	7	8

On all the plots except on Nos. 1 and 5, local varieties were grown; plots 1 and 5 were under Bombay and Peshawar varieties respectively. Seeds for plots 1 to 7 were sown in seed beds in the end of June, and the seedlings were transplanted a month later. Seedlings for No. 8 were transplanted in the end of August, the seeds having been sown a month previous. The first four pickings of ripe fruits from plots 2, 3, 4, 6 and 7 were made on the 30th of October, the 12th of November, the 24th of November and 8th of December. Fruits from plot No. 8 were first picked on the 20th of December.

The yields were low as compared with those normally obtained. This was largely due to over-spacing, the plants being sown $24'' \times 24''$ apart as against the usual $18'' \times 12''$. The soil was also poorer than that ordinarily used for

the chilli crop, and the young plants were attacked by white-ants. The crop was put to a severe test on account of the unfavourable weather. From the middle of September to the first week of October, when the plants had commenced to flower, and consequently were in the stage when they are most readily infected, the days were cloudy and there was more or less rain almost every day. The relative humidity was therefore very high—between 85 and 96 per cent. Those were the conditions highly favourable for causing an epidemic, and there was one, judging from the amount of disease in the control plots and in the ryots' fields. Another factor which tried the efficiency of the control measures was the presence of a second disease, hitherto unobserved on this crop; this was due to *Choanephora Cucurbitarum* (B. & Rav.) Thaxt.[1] which caused much damage. It started from the flower or leaf-bud and led to a wet rot of the shoot.

In spite of these adverse circumstances, one of the control measures, *viz.*, spraying, gave satisfactory results.

To judge the effects of spraying and manuring on the disease on fruits we need only consider the results of the first four pickings done up to the 8th of December, because after this date the percentage of diseased fruits was found to be negligible.

	Weight of black *healthy* fruits		Weight of fresh *diseased* fruits		Total weight of fresh fruits		Percentage of diseased fruits					The date when the disease was first observed
							1st picking on 30th October	2nd picking on 15th November	3rd picking on 24th November	4th picking on 8th December	Total percentage of diseased fruits	
	lb.	oz.	lb.	oz.	lb.	oz.						
(1) Bombay variety	..	..	..	..	..	..	..	..	..	..	..	15th October.
(2) Cuttack variety	25	13	9	12	35	13	68·8	22·0	44·0	7·5	27·4	27th September
(3) Control	38	6	20	8	58	14	78·5	30·0	33·0	12·8	34·3	5th October.
(4) Manure I	207	0	91	10½	298	10½	2·0	17·5	25·4	17·3	19·8	8th October
(5) Pusa variety	..	..	..	..	..	..	..	..	..	..	..	..
(6) Control	104	12	57	5½	162	1	72·8	33·3	37·0	16·6	35·4	8th October.
(7) Sindhuri	120	8	40	7½	160	15	11·6	7·5	10·0	7·5	7·4	10th October.
(8) Local variety	..	..	..	..	..	..	..	..	..	..	..	26th October.

[1] Thaxter, R. L. "*Choanephora Cucurbitarum* (B. & Rav.) Thaxter on Chillies," *Ann. Bot.*, XXXIV (1920), p. 329.

Plot Nos. 1 and 5. As the *Vermicularia* disease is very common on varieties of chillies grown locally, seeds from Bombay and Peshawar, where this disease has not as yet been reported from, were tried in order to see if plants raised from them were disease resistant. Unfortunately these varieties did not do well here ; the growth of the plants was stunted and the yield of fruits negligible ; the Bombay variety suffered badly from leaf-curl as well.

Plot No. 2. In fields lined by a row of trees those plants that grow within the shadow-limits of those trees have been found to be healthy, while those planted outside the shadow-limit get badly attacked by die-back. Small wayside chilli plots surrounded by huts or by big trees have been observed to be free from the disease when other plots a little further away but in the open become infected.

The reason why plants growing under shade remain healthy while those in the open are affected is, that moisture plays a very important factor in the development and spread of the disease. In October and November night dews and ground fogs are heavy and the plants consequently become dripping wet in the night and remain so for some time after sunrise ; this high humidity is favourable for the spread of the disease. But under shade there is very little mist or dew-fall, and consequently here the atmosphere is comparatively dry, dry enough to check the spread of the disease.

Inoculation experiments under laboratory conditions have shown that a great deal of moisture is required for the inoculations to succeed and for the infection to spread further. If a successfully inoculated plant or fruit is removed to dry surroundings the infection does not progress. Thus laboratory study has confirmed field observations.

As a result of these observations another line of control suggested itself. It seemed probable that if chillies were grown as a mixed crop with maize or some such other economic crop, which grows rapidly and gives a good shade, the chilli crop would perhaps remain healthy or would be very little affected.

For this reason the local varieties of chillies were grown mixed with maize. Drills of maize were laid down after every three rows of chillies. Maize was sown in drills in the end of July when the chilli seeds were sown in seed beds. The selection of maize as a mixed crop was unfortunate ; it flowered and cobbed in September and in the beginning of October it began to turn brown ; again in the first week of this month it was blown over in the high winds and damaged the chilli plants. It was consequently uprooted two or three weeks later.

The chilli plants were rather smaller in size, flowered later and gave comparatively smaller yield of fruits than those of the control plots. They

also showed a greater amount of leaf curl. However the effect of shade as far as the disease on the chilli stems was concerned was very marked. They suffered the least from die-back. That shade does check die-back is also evident from the following observation. It so happened that the three end lines of chillies had no drill of maize to their south and so they remained unshaded. The result was that this set of three lines was more badly attacked than the rest. It had 32 plants suffering from die-back; the neighbouring set of three rows had 11 diseased plants, while in the remaining 9 sets the number of died-back plants varied from 6 to 9 each set. It was in the unshaded rows here the disease first appeared.

Whether shade has any effect on the total yield and on the percentage of diseased fruits cannot be judged from this experiment because, as already stated, maize plants were blown over by high winds in the beginning of October and they were uprooted three weeks later, as instead of doing any further good they were damaging the chilli plants underneath them. Thus at the time when the fruits generally get attacked they remained unshaded. The percentage of diseased fruits in each of the first four pickings was 68·5, 22·0, 11·0 and 7·7, almost the same as that for the control plots.

Plot Nos. 3 and 6. These were control plots. Of all the plots, plot No. 3 was most severely attacked by *Choanephora* and die-back; the natural result was that the yield of fruits was greatly reduced; the attack was first observed on the 5th of October. Plot No. 6 was found attacked three days later. It was a little more diseased than the sprayed plot No. 7, but much less than No. 3. Still however the percentage of diseased fruits in both these control plots was almost the same, and much higher than that in the sprayed plot. The percentage of diseased fruits from each of the first four pickings from plot No. 3 was 78·5, 30·0, 33·0 and 12·8, and from No. 6 was 72·8, 33·3, 37·0 and 16·6.

Plot Nos. 4 and 8. From experiments made in previous years it had been found that chillies sown a month later than usual suffer very little from die-back, but the yield of fruits was greatly reduced. To see if manuring could remedy this defect, plot No. 8 was manured at the rate of 2 cwt. of superphosphate and 1 cwt. of nitrate of soda per acre. Seeds for this plot were sown in seed beds in the end of August, and the seedlings were transplanted a month later. Plot No. 4, which was manured at the same rate, served as a check, the sowing time being normal.

As a result of manuring the growth of the plants in plot No. 4 was more vigorous than those in the other plots. Die-back on this plot was observed first on the 8th of October; and it was rather severe, but was not very much more than on plot No. 6 and decidedly less than that on No. 3. The total

yield of fruits was much heavier than in any of the other plots, and the percentage of diseased fruits was less than that from the control plots, but more than that from the sprayed plot. The percentage for each of the first four pickings being 25·0, 17·5, 25·4 and 17·3.

On plot No. 8 the disease was first observed on the 26th of October. Altogether only 45 plants were found infected by the end of November. This showed once again that a late sown crop easily escaped damage caused by die-back. The fruits were not ready for picking before the third week of December, and therefore naturally the percentage of diseased fruits was negligible.

Plot No. 7. The disease on this plot was first observed on the 10th of October, but the first application of one per cent. Burgundy mixture, at the rate of 125 gallons per acre, was given on the 29th of September as on the 27th one plant in plot No. 2 was found diseased. The mixture happened to be very alkaline, and consequently the tender tips of the flowering shoots got rather badly burnt. The next application was given on the 17th when the fruits had set. It had no bad effect on the plants as the mixture was made very slightly alkaline. The sprayed plants had decidedly less of die-back than the plots 3, 4 and 6, and yielded more healthy fruits. The percentage of diseased fruits for each of the first four pickings was 11·6, 7·5, 10·0 and 7·5. The total yield of fresh fruits from these pickings was less than that of the control plot No. 6, but the yield of healthy fruits was higher. Not only did the freshly picked fruits from the sprayed plot compare favourably in regard to the percentage of disease than those from the unsprayed plots, but they also stood drying better. Healthy fruits picked from the sprayed plot remained healthy on drying, and the skin of the dried fruits was bright ruby red. But fruits from unsprayed plots that were healthy when picked developed diseased spots on drying. It has been found that *Gloeosporium* on plantains[1] and on other fruits and leaves[2] is capable of lying dormant till there are suitable circumstances for developing its activities ; similarly *Vermicularia* may lie dormant on the pods and resume its growth in the moist and warm conditions of the drying heap ; of course, the attack of the disease on the drying pods is not very severe, but the spotted and discoloured skin of the dry fruits is bound to lessen the market value.

The reason why only two sprayings were given is that before the third could be applied there was a sudden fall of temperature and of the humidity percentage

[1] Dastur, J. F. Spraying for Ripe Rot of the Plantain Fruit. *Ur. Agric. Indica*, XI, 1916, p. 145.

[2] Shear, C. L., & Wood, A. K. Studies of Fungus parasites belonging to the Genus *Glomerella*. *U. S. Dept. of Agric. Bur. of Pl. Ind. Bull. No.* 252, 1913, p. 95.

the beginning of November and the disease was consequently checked. The disease did not progress further down the infected part of the plants, and no fresh infections on the plants were observed after the cold weather. That the disease was checked is also evident from the fact that in the first picking the percentage of diseased pods in the plots 3, 4 and 6 was 78·5, 25·0 and 72·8 respectively, while in the second picking the percentage fell to 30·0, 17·5 and 33·3 for the respective plots. It was further found that better results are obtained if the diseased parts of the plants are thoroughly pruned off before spraying, as the fungicide is applied only as a preventive measure and not as a curative. The fungus hyphæ once inside the tissues are little harmed by the application of any spray; therefore the spread of the fungus from within the diseased tissues to the neighbouring healthy parts and the ultimate death of the plant cannot be prevented; the spray only checks new infections from outside. However, it is doubtful if the pruning of diseased shoots is practicable on a field scale.

From the above experiments the following deductions can be drawn:—

1. That the disease appears after the end of the rains—in the first and second week of October; that it attacks plants only at a definite stage in their growth, *i.e.*, when the flowers have set, and that it disappears as soon as the cold weather commences in the beginning of November; the disease on the plant is, therefore, of a very short duration. Fruits that mature before the beginning of December are much damaged by the disease, but the percentage of infection on fruits that ripen later is negligible. These observations are completely in accord with those made in the ryots' fields. It seems, therefore probable that if a late maturing variety could be successfully grown in Bihar it would escape the disease.

2. That Bombay and Peshawar varieties do not grow well in Bihar.

3. That shaded plants suffer less from die-back and *Choanephora* than unshaded; no conclusions can be drawn regarding the effect of shade on the yield of fruits and percentage of disease.

4. That the application of 2 cwt. superphosphate + 1 cwt. nitrate of soda per acre not only increases the total yield of fruits, but also reduces the percentage of diseased fruits. It is doubtful if the use of artificial manures is possible on account of the present inflated prices of these fertilizers.

5. That two applications of one per cent. Burgundy mixture are enough to control the disease, both on the plants and on the fruits. It is possible that only one application, if given at the right time, may be equally efficacious.

6. That healthy fruits from the sprayed plants remain unspotted on drying, while those from the unsprayed plants develop the disease.

7. That perhaps better results may be got by a combination of application of manure and spraying with Burgundy mixture.

8. That the late sown crop suffers little from die-back and *Choanephora*.

These experiments with a few additions and alterations were repeated in 1918, 1919 and 1920, in order to confirm the results obtained in 1917, but this object was not attained because in these years there was no incidence of this disease, not only at Pusa, but also in the chilli-growing tracts north of the Ganges. It has already been pointed out that high humidity is correlated with the incidence of the disease. These three years, 1918, 1919 and 1920, were comparatively drier and warmer than the previous three years, at the time when the plants are susceptible to infection. Judging from the observations of the last six years it seems that this disease in Bihar becomes virulent when the humidity percentage in the second half of September (when the plants begin to flower) is on an average above 85.

Summary.

The die-back disease of chillies in Bihar, caused by *Vermicularia Capsici* Syd., is described.

The disease first appears in the end of September or in the first half of October when the plants have commenced to flower.

Plants growing in the shade of trees escape the infection.

The late sown crop is also free from this disease.

The infection as a rule commences from the growing point or the flower-bud. The stem dies back as the infection spreads downwards.

The attack on the stem is checked with the first onset of the cold weather.

Infected fruits become spotted when they are ripe, but fruits that mature after the beginning of December are generally free from disease.

Seeds of diseased fruits may become infected. The hyphæ are found not only on the seed coats but also in the inner tissues.

The germination of the infected seed and the development of the seedling depend on the extent of the diseased condition of the seed.

Cultural characters of the fungus are described.

Inoculation experiments show that the plants take the infection only when the humidity is very high. If it is reduced below a certain limit progress of the infection is checked.

From field observations of the last six years it appears that this disease attains great virulence when the humidity percentage is on an average above 85 in the latter half of September.

Control measures tried in 1917 show that Burgundy mixture is useful in checking the disease.